Angioplasty of an Idiopathic Intracranial Arterial Stenosis

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Summary

Treatment of symptomatic intracranial atherosclerotic stenosis usually involves maximizing medical therapy. In patients with medically refractory symptoms despite maximum pharmacologic therapy, intracranial angioplasty and/or stenting has become an accepted treatment option. The use of percutaneous transluminal angioplasty (PTA) for idiopathic intracranial stenoses has not been reported to date. We describe a case of idiopathic intracranial stenosis which was refractory to medical therapy and was successfully treated with percutaneous transluminal angioplasty. The presenting symptoms included multiple episodes of aphasia and right-sided weakness as well as a left basal ganglia infarct. The patient underwent treatment with two intracranial angioplasty procedures. There was a recurrence of the stenosis and symptoms following the first procedure. However, after a second treatment with a slightly larger balloon, flow in the MCA normalized. Furthermore, the symptoms attributed to her MCA stenosis had essentially resolved. This case suggests that patients with medically refractory idiopathic intracranial stenosis can be successfully treated with percutaneous transluminal angioplasty.

Introduction

Angioplasty is increasingly used to treat symptomatic intracranial arterial stenoses. There are numerous reports describing the use of PTA with or without stenting in the treatment of atherosclerotic disease and vasospasm associated with subarachnoid hemorrhage [1-3]. However, to our knowledge, the use of angioplasty to treat inflammatory or idiopathic intracranial stenosis has not been reported to date. We speculate that concern regarding the integrity of the wall of the stenosed arterial segment and the potential for iatrogenic vessel dissection or rupture may have limited the use of angioplasty in these patients. We present a case of idiopathic proximal M1 segment middle cerebral artery (MCA) stenosis that was successfully treated with PTA.

Technical Report

Clinical History. A 21-year-old woman presented with acute onset of transient right-sided hemiparesis and expressive aphasia. An MRI/MRA revealed an acute left basal ganglia infarct and a high-grade stenosis of the left proximal M1 MCA segment. She was admitted to the hospital and was started on aspirin and heparin. Despite intensive inpatient medical therapy, she continued to have multiple left hemispheric transient ischemic attacks (TIAs) with aphasia and extremity weakness. Evaluation of the serum and cerebrospinal fluid (CSF) was negative, without evidence of abnormal inflammatory markers. The CSF examination was also negative for infectious etiologies, including viruses. She was evaluated by the Neurosurgery and Interventional Neuroradiology Depart-

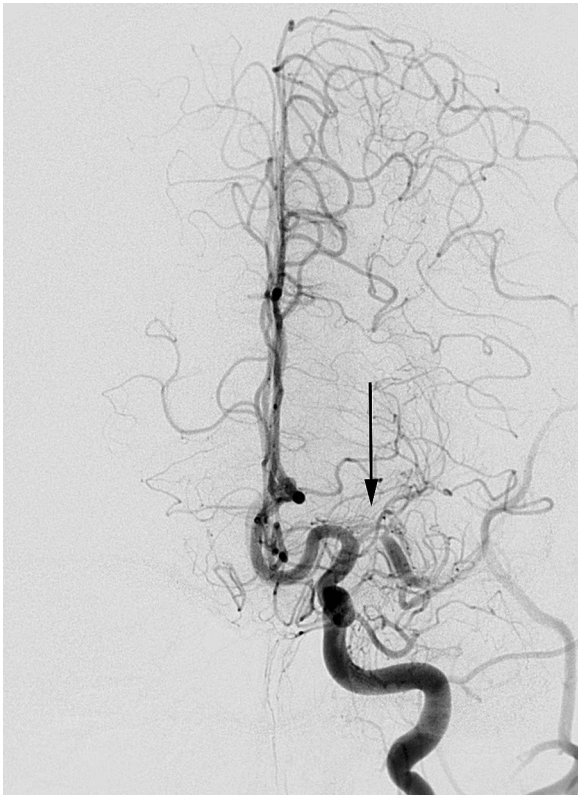

Figure 1 Left ICA angiogram demonstrates a high-grade stenosis of the proximal M1 segment (arrow) with poor antegrade flow in the distal MCA territory. There is a shift of the ACA-MCA watershed with ACA-MCA pial collaterals present.

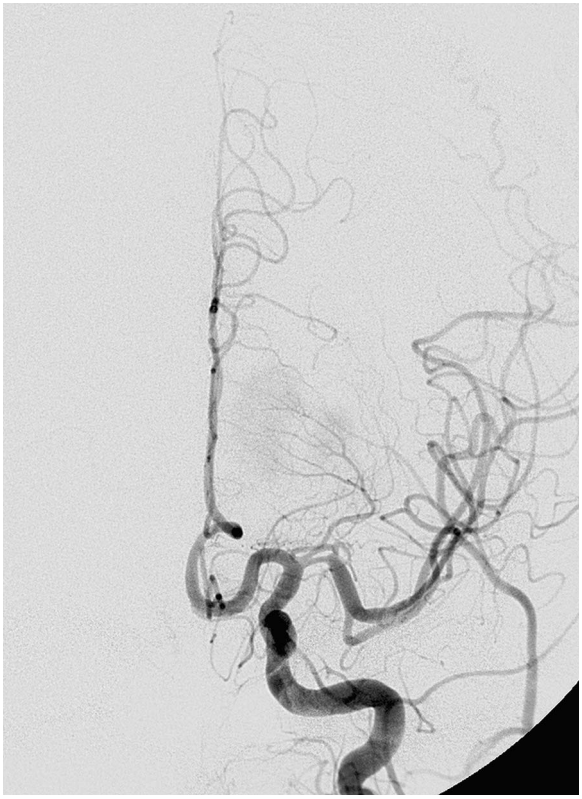

Figure 2 Following the initial angioplasty treatment with a 1.5 mm diameter balloon, there is a reduction in the M1 stenosis and improved flow in the distal MCA territory. The ACA-MCA watershed area has returned to normal.

ments for possible surgical or endovascular treatment options. A four-vessel cerebral angiogram confirmed a high-grade left M1 stenosis with associated left anterior cerebral artery (ACA) to MCA pial collaterals (figure 1). Antegrade flow in the MCA distal to the stenosis was poor. Due to persistent TIAs despite optimal medical management, it was elected to proceed with intracranial angioplasty.

Intervention. Informed consent was obtained for the procedure and the patient was placed under general anesthesia by the Anesthesia Department. After gaining arterial access, systemic heparinization was maintained to keep the activated clotting time (ACT) near 300 seconds. A 5Fr guiding catheter was placed in the left internal carotid artery (ICA). Because of the concern over potential vessel rupture, we initially chose to use a small caliber balloon. Over a Transcend 14 microguidewire (Boston Scientific, Fremont, CA), a Voyager 1.5 mm x 12 mm angioplasty balloon (Guidant Corporation, Indianapolis, IN) was advanced across the stenosis and carefully inflated to a nominal pressure of eight atmospheres. The balloon was deflated and removed. A post-angioplasty left ICA angiogram was then performed and demonstrated improvement in the vessel caliber and improved antegrade flow in the left MCA territory (figure 2). The patient was observed in the neurologic intensive care unit (NICU) for four days and discharged on 325 mg aspirin per day and 75 mg clopidogrel bisulfate per day without any further neurologic events.

One month following discharge the patient presented with multiple transient episodes of right upper extremity weakness, but she had no fixed neurologic deficit. A repeat cerebral angiogram was performed and revealed a restenosis of the left M1 MCA segment with delayed antegrade flow in the left MCA territory (figure 3). The degree of stenosis was slightly less severe than the presenting angiogram per-

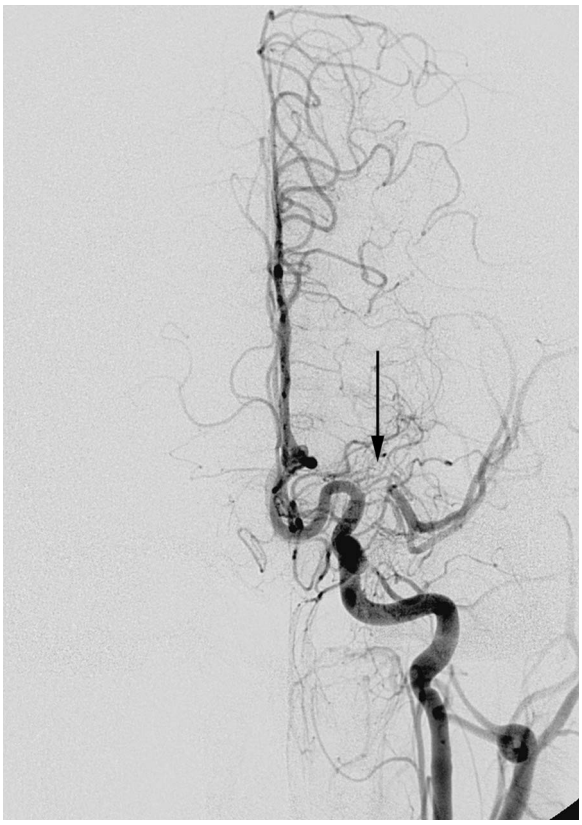

Figure 3 Left ICA angiography one month following angioplasty demonstrates a recurrent high-grade stenosis of the proximal MCA (arrow).

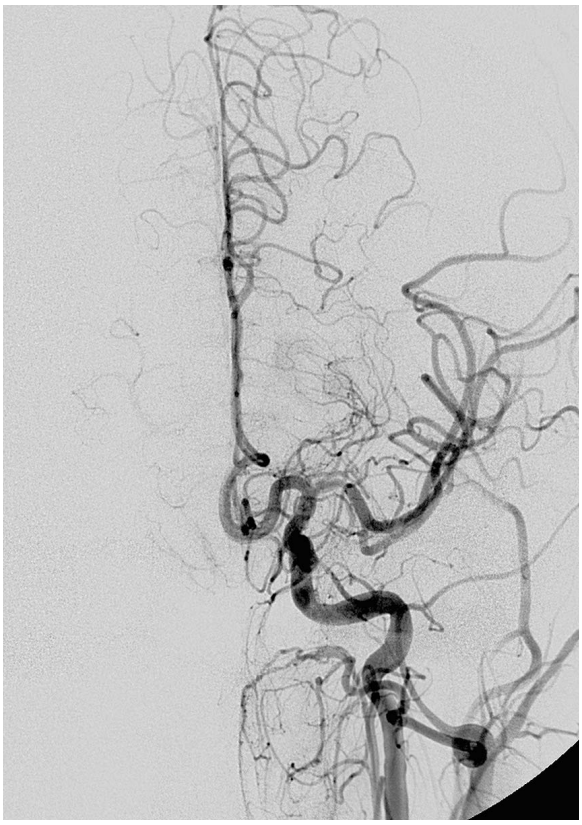

Figure 4 Following angioplasty with a 2.5mm diameter balloon, there is improved flow in the distal MCA territory and a significant reduction in the degree of MCA stenosis.

formed one month previously. A 6Fr guiding catheter was placed in the left ICA and systemic heparinization was again instituted. Given the recrudescence of symptoms and stenosis since the first treatment, we chose to increase the balloon diameter. A Voyager 2.5 mm x 8 mm angioplasty balloon (Guidant Corporation) was advanced across the stenosis over a Transcend 14 soft tip microguidewire (Boston Scientific). The balloon was inflated to a nominal pressure of eight atmospheres and deflated. Following removal of the balloon, a left ICA angiogram was performed and revealed improved flow in the MCA territory and improvement in the degree of M1 stenosis (figure 4). Following observation in the NICU overnight, she was discharged on the same regimen of aspirin and clopidogrel without further neurologic symptoms. A Diamox brain perfusion study was performed 30 days after the second angioplasty procedure and revealed no inducible perfusion defects. Follow-up catheter angiography 11 weeks after the second angioplasty procedure revealed minimal residual stenosis of the left M1 origin and there was normal flow in the left MCA territory (figure 5). A computed tomography angiogram (CTA) performed eight months after her initial presentation demonstrated a stable appearance of the proximal M1 artery with only minimal narrowing (figure 6). One month prior to the CTA examination, the patient experienced a TIA localized to the right hand that lasted one to two minutes. This TIA occurred as her antiplatelet therapy was changed from clopidogrel to ticlopidine for hives attributed to clopidogrel. Otherwise, she has had no neurologic events.

Discussion

The medical management for atherosclerotic intracranial stenosis has been described and traditionally included the use of warfarin[4,5]. However the results of the WASID trial de-

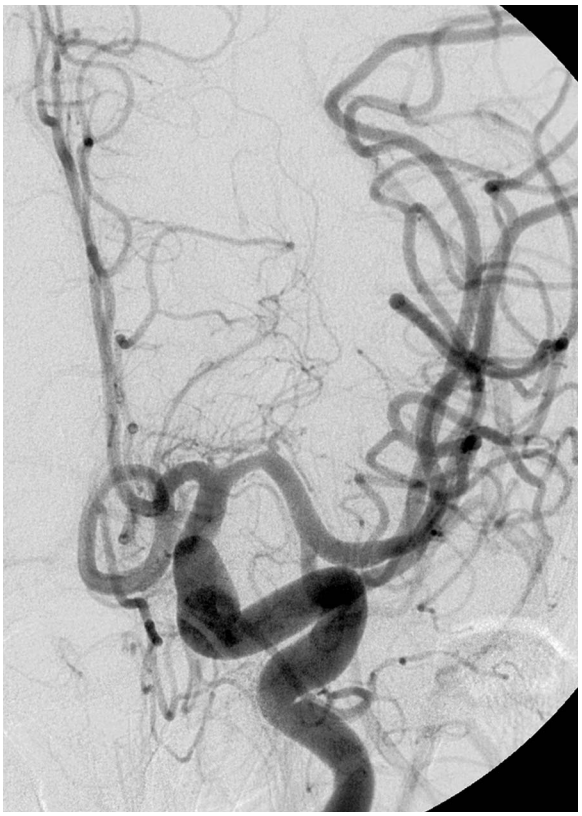

Figure 5 Left ICA angiography 11 weeks after the second angioplasty procedure demonstrates a minimal narrowing of the left MCA.

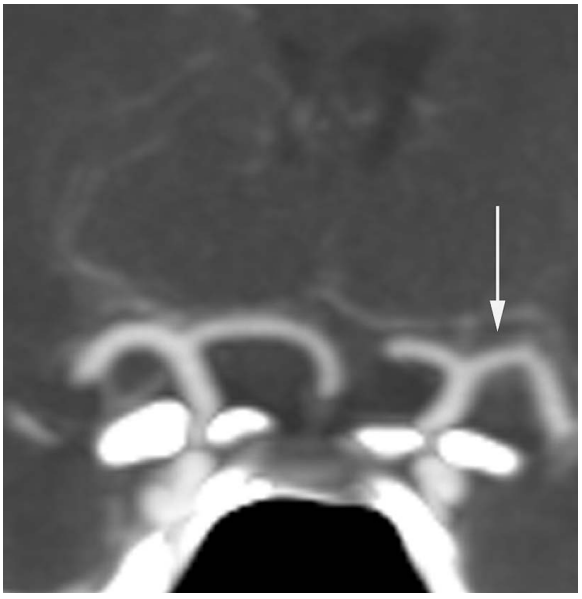

Figure 6 CTA 8 months after initial presentation reveals a stable appearance of the left M1 MCA with only minimal narrowing at the site of previous angioplasties (arrow).

monstrated no added benefit for warfarin compared with aspirin and these lesions are now generally treated with aspirin and/or clopidogrel [6]. Surgical options such as STA-MCA bypass procedures have shown limited benefit for MCA stenoses [7]. Intracranial angioplasty is an accepted treatment option for symptomatic atherosclerotic stenoses that are refractory to maximum medical therapy [8]. Because of technical advancements in the field of interventional neuroradiology, it is now possible to navigate coronary angioplasty balloons into the intracranial circulation. Guided by their experience with angioplasty for the treatment of vasospasm, operators have begun to apply this technique to the treatment of atherosclerotic lesions.

A crucial difference between atherosclerotic and inflammatory stenoses is the integrity of the arterial wall. The plaque in an atherosclerotic lesion is intentionally disrupted by the angioplasty balloon. While it is possible to cause vessel dissection and rupture in these cases, the surrounding arterial wall is usually sufficiently intact to allow for purposeful balloon dilation. In cases of significant iatrogenic dissection, subsequent placement of a stent can be performed. In contrast to atherosclerotic lesions, the integrity of the arterial wall is often in doubt in inflammatory or idiopathic stenoses. With current imaging techniques, the integrity and composition of the arterial wall involved cannot be determined. There is a concern that the vessel wall may be significantly friable and any attempts at endovascular treatment should be performed cautiously. For this reason, the initial treatment should be aimed at maximizing medical therapy.

In the case described above, the etiology of the MCA stenosis is unknown. Potential causes include early moyamoya disease, vasculitis, sarcoid or low-grade CNS infection, but no cause could be determined despite an intensive workup. Although possible, the lesion did not appear to represent a simple dissection. Angioplasty was only entertained after the patient failed maximal medical therapy. A brain perfusion study was not performed prior to intervention because of the obvious severe stenosis and angiographic evidence of inadequate MCA territory flow.

In the first angioplasty session, we purposely used an undersized balloon to limit the pressure on the vessel wall. After the patient re-

turned with a recurrent stenosis, the balloon was up-sized by 1 mm and this resulted in a satisfactory result. There is a potential risk for vessel rupture or dissection when using a 2.5 mm balloon in a small MCA. However, we felt that use of a 2.5 mm balloon was needed for the second procedure due to the rapid recurrence of stenosis. The long-term durability of this treatment is uncertain, especially in a patient this young, and it is possible that further angioplasty treatments will be required. There are new stent devices under development for use in the intracranial vasculature that are easier to deploy than traditional coronary stents[9,10]. However, the use of a stent in a 21-year-old patient would be worrisome. The long-term patency rates for intracranial stents are unknown and their indications are undefined as of yet. As in our case and all intracranial angioplasty procedures, operators must be prepared to place a stent in the event of a significant balloon induced dissection or rupture. While it is true that drug eluting stents may prove to be a viable treatment for young patients with intracranial stenoses, the long term outcomes for these devices are still uncertain.

The implications of this case are limited because it represents a single case report. However, as demonstrated in our patient, gentle angioplasty in cases of medically refractory idiopathic intracranial stenosis may allow practitioners to bridge the time until the vessel heals on its own and hopefully prevent the need for an intracranial stent.

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